

ROLE OF ANIMAL MEATS IN CARDIOVASCULAR DISEASES WITH REFERENCE TO SUB-SAHARAN AFRICA: A REVIEW

Lokuruka MNI*1



Michael Lokuruka

*Corresponding author email: lokuruka@hotmail.com

¹Senior Lecturer in Food Science, Department of Dairy and Food Science and Technology, Egerton University, P.O. Box 536-20115, Egerton, Kenya



FILCAN JOURNAL OF FOOD AGRICULTURE

ABSTRACT

Although animal food products are important sources of dietary protein in the diets of many African communities, questions are being raised concerning the role of animal meats and other animal products in chronic diseases including cardiovascular diseases. This review of the literature was, therefore, conducted to provide information on the role of animal meats and their nutrients in cardiovascular diseases, with reference to sub-Saharan Africa. In sub-Saharan Africa, infectious diseases are the current commonest cause of death, but cardiovascular diseases are set to surpass them as the major cause of mortality and morbidity. Although rare in the past, there are indications that coronary heart disease will become a burden to the health care system in sub-Saharan Africa. Diet is an important part of the control and management of cardiovascular diseases as most risk factors including diabetes mellitus, obesity, low-density lipoprotein and triacylglycerol levels, alcohol and blood pressure are all connected to diet. Animal meats can be significant sources of dietary saturated fatty acids and cholesterol due to their considerable lipid content. Dietary saturated fatty acids and cholesterol are significant influences on the development and progression of atherosclerosis, a major type of cardiovascular disease. Heart disease and high serum cholesterol also increase the risk of stroke. Although dietary protein is considered to be of minor importance in the aetiology of cardiovascular disease, epidemiological studies have shown the consumption of animal protein to be positively correlated with cardiovascular disease incidence, while that of vegetable protein is negatively correlated with cardiovascular disease mortality rates. Also, due to the higher content of cholesterol in organ meats compared to skeletal muscles, it is recommended that their dietary consumption be limited. It, therefore, seems beneficial to reduce the dietary intake of saturated fatty acids and cholesterol from meats and other high-fat, high-cholesterol animal foods, except fatty fish, since it is established on the basis of documented scientific evidence that reduction in serum cholesterol results in reduction of coronary morbidity and mortality. This review, therefore, recommends reduction in the intake of fatty red animal meat, while promoting consumption of portion-controlled lean white meats such as poultry, wild game and fatty fish. This can be achieved by integrating these recommendations into food and nutritional policy practice in Africa.

Key words: Meats, CVD risk, sub-Saharan Africa

UTICAN JOURNAL OF FOOD AGRICULTURE

Cardiovascular Diseases (CVD)

Cardiovascular diseases, which include any disease of the heart or blood vessels, are major global causes of morbidity and mortality. The main types of CVD are coronary heart disease (CHD), stroke and atherosclerosis. Coronary heart disease results from accumulation of atheromatous plaques within the walls of the arteries that supply the myocardium [1]. Coronary heart disease is synonymous with ischaemic heart disease, and may be manifest by angina (chest pain), or acute myocardial infarction (AMI) (heart failure) [1]. When coronary arteries become clogged by fat and cholesterol deposits and cannot supply enough blood to the heart, CHD results.

Volume 10 No. 1

January 2010

A stroke is an interruption of the blood supply to any part of the brain. Ischaemic and haemorrhagic stroke are the two major types of stroke [1]. High blood pressure is the major cause of stroke [2]. The risk of stroke is also increased by age, family history of stroke, smoking, diabetes, high serum cholesterol, and heart disease [1]. Also, nutritionally-related questions about fat and oil consumption continue to be raised due to the disease condition known as atherosclerosis, in which plaques of fatty acids and cholesterol are deposited on arterial walls. These plaques are dangerous because they restrict blood flow, and when they loosen, they can form emboli (blood clots) that may lodge in a coronary artery and cause a heart attack [1]. Epidemiological evidence points to the promotion of atherosclerosis by high serum concentrations of saturated dietary lipids and cholesterol [1].

Cardiovascular diseases currently cause an estimated 17 million deaths/year globally, which are projected to reach 25 million by 2020 [3]. Among CVD, CHD is the leading cause of death and disability in both men and women. In those under age 65, death rates from CHD are substantially higher for men than for women. After age 65, death rates for both genders become similar [4]. Cardiovascular diseases are responsible for 30% of all annual deaths in all regions of the world [5].

General Risk Factors for CVD

The non-modifiable risk factors for CVD are increasing age, gender, heredity, while the major identified modifiable risk factors are cigarette smoking, high blood pressure, elevated plasma cholesterol, obesity, diabetes and elevated plasma triacylglycerols (TAGs) [6]. Diet is an important part of the control and management of CVD; diet involves a modifiable set of factors, which if well managed can reduce the risk of CVD. Most risk factors including diabetes, obesity, low-density lipoprotein (LDL) levels, triacylglycerol levels, alcohol and blood pressure are all connected to diet. Changes in diet alone without control and improvement of other risk factors cannot, however, prevent CVD [6].

Food fatty acids (FAs) mainly comprise saturated fatty acids (SAFA), monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA). The PUFA mainly include n-3, n-6 and n-9 FAs, represented by linolenic, arachidonic and oleic acid, respectively. Cholesterol performs important roles in the body despite its



adverse association with atherosclerosis [7]. The American Heart Association (AHA) recommends limiting dietary cholesterol to <300 mg/day due to its role in atherosclerosis [8]. However, the notion that cholesterol causes atherosclerosis in humans or that reduction in the consumption of cholesterol foods to the exclusion of other factors reduces the incidence of this disease is still contentious, often due to the use of epidemiological data from animal experiments and extrapolating the conclusions to humans. Hyperlipidemia, which is associated with elevated serum cholesterol, cholesteryl esters and TAG, has been identified as a major risk factor for atherosclerosis. High-density lipoproteins (HDL) carry cholesterol back to the liver from the bloodstream and are thought to be protective by taking the extra cholesterol out of the blood. Low-density lipoproteins (LDL) transport cholesterol through the blood to the cells and usually comprise most of the blood cholesterol. Very lowdensity lipoproteins (VLDL) also keep cholesterol in circulation and may contribute to atherosclerosis. The LDL, total cholesterol/HDL and/or the LDL/HDL ratios are often assumed to be relative measures of CVD risk. A ratio of LDL/HDL above 5.0, total cholesterol, LDL, and triglyceride levels >200, 150, and 150 mg/dL, respectively, and HDL <35 mg/dL are associated with elevated CVD risk [9].

It is established that individuals with premature CVD exhibit some or all of the following: a) increased LDL levels; b) increased cholesteryl esters and TAG, primarily as VLDL; and c) increased TAG with normal cholesterol, primarily as VLDL [10]. Prospective studies have found a positive correlation between serum LDL and atherosclerotic events, and this appears to be inversely related to serum HDL [11]. The effect of HDL, however, does not fully explain CVD mortality differences between populations. Also, despite the cardio-protective effect of HDL and the widespread acceptance of the role of LDL-cholesterol (LDL-C), it remains true that many patients suffering from myocardial infarction have relatively normal levels of LDL-C [11]. Also, there is still ongoing debate as to whether high LDL-C or low HDL-cholesterol is the better predictor of CVD risk.

REVIEW

CVD Risk in Sub-Saharan Africa

Although infectious diseases are the commonest cause of death in sub-Saharan Africa (SSA), there are indications that CVD will eclipse infectious diseases within a few years [12]. In Kenya, HIV/AIDS claims 100 000-150 000 lives annually compared with over 42 000, 30 000 and 20 000 by CVD, malaria and tuberculosis, respectively [3]. In 2002, cerebrovascular disease, ischaemic stroke and other CVD were responsible for 32, 37 and 31%, respectively of 20 000 deaths attributed to CVD in males, and 37, 28 and 36%, respectively of 24 000 female deaths in Kenya [3]. Ischaemic stroke and cerebrovascular disease were the main CVD sub-type killers of Kenyan males and females, respectively.

In developing countries, there are indications that deaths from CVD are rising with changes in lifestyles, "modernization," urbanization and occupational sedentarization [3].



Volume 10 No. 1 January 2010 ISSN 1684 5374

AFRICAN JOURNAL OF FOOD AGRICULTURE

In a study involving 8581 rural Tanzanians, Swai et al. [13] assessed the prevalence of CHD risk factors. The subjects aged 15 and above were spread out in 8 villages in 3 regions in rural Tanzania representing a range of different socioeconomic deprivation. The main outcome measures examined were serum cholesterol and TAG level, prevalence of dyslipidaemia, hypertension, smoking, overweight, impaired glucose tolerance (IGT) and diabetes; electrocardiographic (ECG) changes in subjects pointing to the likelihood of ischaemic stroke were also examined. The results showed the mean serum cholesterol levels in men were 4.2, 3.4 and 3.7 mmol/L, and in women 4.4, 3.6 and 3.9 mmol/L in Kilimanjaro, Morogoro and Mara regions, respectively. In Kilimanjaro region, 17.4% of men and 19.0% of women had values greater than 5.2 mmol/L (>200 mg/dL) compared with 5.0 and 6.7% of men and women, respectively, in Morogoro region, and 4.8 and 6.9% of men and women, respectively, in Mara region. Systolic and diastolic pressure increased with age in both men and women in the 3 regions, with the most marked in the Kilimanjaro region and the smallest in Mara region. Hypertension was found in 6.6% of men and 7.5% of women in the Kilimanjaro region, 3.3 and 4.7% in Morogoro, and 2.6 and 3.4% in Mara region. Smoking was found in 42.6% of men in the Kilimanjaro region, 28.2 and 8.6% of men in Morogoro and Mara, respectively. Less than 4% of women in the 3 regions smoked. Only 2.4-4.0% of men and 7.9-10.5% of women were obese. Changes in ECD indicating risk of developing CVD were recorded in 2.7-7.8% of men, 3.7-17.4% of women <40 years of age. Diabetes was detected in 0.6-0.8% of subjects. Also, the proportion of men with ≥ 2 risk factors for CHD ranged from 0.2% in Mara to 4.6% in the Kilimanjaro region. For women, the corresponding figures were 0% in Mara and 1.1% in the Morogoro region. The study also showed that the proportion increased in those with IGT (9.3%) and diabetes (14.1%). Similarly, those with ECD signals showing the likelihood of ischaemic disease had a two-fold increase in risk factors. From the study, it was evident that cardiovascular risk factor levels made it unlikely that time that CHD would emerge as a significant problem among rural Tanzanians in the near future.

However, a study aimed at detecting the prevalence of hypertension in an urban and rural area of Tanzania 7 years later [14], was based on two linked cross-sectional population-based surveys in a middle-income district of Dar es Salaam (with 715 adults, >15 years of age) and a village in a relatively prosperous rural area of Kilimanjaro (with 928 subjects). The study established that hypertension prevalence (blood pressure >140 and/or 90 mm Hg, or known hypertensives?? receiving antihypertensive treatment) was 30 and 29% in men and women, respectively, in the Dar es Salaam suburb, and 32% in both men and women in the rural Kilimanjaro area. In both areas, <20% of the hypertensives seemed aware of their diagnosis, with about 10% on treatment, and <1% were controlled (blood pressure 140/90 mm Hg). The study showed there was a high prevalence of hypertension in both rural and urban areas of Tanzania, with low levels of detection, treatment and control. Although no indications were given relating the findings to animal meats consumption by the respondent populations, it is documented that milk consumption was higher in the urban areas (31 L per capita per annum or 85 mL per head per day) than in the rural areas (22 L per head per year or 60 mL/day per head [15]. Areas such as Mara and





Morogoro which were found to have lower CVD risk factors than Dar es Salaam, had the lowest milk production and consumption [16]. The Tanzania milk *per capita* supply in 1988 was 22 L, which had not changed by 1993 (23.2L) [16]. In the 1993 survey, Arusha, Moshi and Kilimanjaro areas had a *per capita* milk supply of 45 L as compared to 6 L and 9.3 L in Dar es Salaam and Mara, respectively [16], although Dar es Salaam received most of its supply from the peri-urban and far away rural areas. Nevertheless, the authors did not link their findings to milk and dairy products consumption in the regions concerned. However, some link between the prevalence of hypertension and a lifestyle of prosperity in both the rural and the urban areas of Tanzania was evident from the study.

In a study in the Gambia [17), the prevalence of hypertension and diabetes was assessed in a 1% population sample of 6048 adults >15 years of age. The study found that 10% of the subjects were hypertensive according to WHO criteria (a diastolic pressure of 95 mm Hg or above and/or a systolic pressure of 160 mm Hg or above). By less conservative criteria (blood pressure of 140/90 mm Hg), 24% of subjects studied were hypertensive. This figure although lower, compares with the 29-32% range of hypertensives in the Tanzania study [14]. Similar to the Tanzania study, the prevalence of hypertension was similar in the rural and urban Gambian communities studied. The study also established that age and obesity were risk factors for hypertension, while the female sex was an additional risk factor for diastolic hypertension, possibly because obesity was associated more with the female sex. Diastolic hypertension is associated with an increased risk of CVD. Obesity was also associated with increasing age, the urban environment, non-manual work and diastolic hypertension. It was noted therefore that hypertension and related non-communicable diseases such as CVD are prevalent in the Gambia. Although, the authors did not examine the link between the prevalence of the CVD associated risk factors with the consumption of animal products, it has been reported that Gambians obtain more than 40% of their daily protein requirements from fish [18]. The country has a milk and dairy products *per capita* consumption of 8 kg [19].

Elsewhere, in a study of heart disease in school children in rural Kenya using a colour-flow echocardiograph, Anabwani and Bonhoeffer [20] examined 1115 children and indicated that the prevalence rate of rheumatic heart disease (RHD) and trivial mitral regurgitation (TMR) was 3 and 6%, respectively. It was apparent that the prevalence of RHD and TMR was surprisingly higher than previously believed, although the findings were not linked by the authors to the consumption of animal meats and other high-cholesterol animal products, or the socio-economic groupings to which parents of the children belonged.

A review of cardiovascular complications of diabetes in SSA through a Medline search of published data over the past 2 decades by Kengne *et al.* [21], revealed that diabetes and related CVD complications though considered rare in Africa, are on the rise, and, are regularly associated with classic cardiovascular risk factors. The literature search revealed that CHD may affect 5-8% of type 2 diabetic patients and up to 5% of diabetic patients present with cerebrovascular accidents at diagnosis, while





peripheral vascular disease prevalence seemed to vary across sites from 4-28%. The search showed that diabetes and related CVD complications are gaining in importance in SSA, although the relative contribution of putative risk factors was not well defined.

In a recent study, where Steyn *et al.* [22] assessed the impact of modifiable CVD risk factors on myocardial infarction (MI) in 9 countries of sub-Saharan Africa, five risk factors (smoking history, diabetes history, hypertension history, abdominal obesity and ratio of apolipoprotein B to apolipoprotein A-1) provided a population attributable risk of 89% for AMI. The risk of AMI rose with higher income and education in the black African groups in contrast to the findings in the other African groups. A history of hypertension revealed higher MI in the black African group. The study showed that CVD risk factors account for about 90% of MI observed in the African populations in the countries studied (South Africa, Botswana, Mozambique, Nigeria, Kenya, Zimbabwe, Benin, Cameroon and Tanzania).

Consumption of some animal proteins with reference to Kenya

In 2004, the annual *per capita* milk consumption in Kenya was 113 L, while the corresponding cholesterol intake was 89 mL [23]. The sub-Saharan Africa *per capita* supply of milk is documented at 27 L [24] and West Africa, Central Africa, East Africa and Southern Africa regions had a *per capita* supply of 13.2, 9.2, 56.1 and 19.2 L, respectively [24]. All these regions do not fully meet their milk and dairy products requirements from internal production but try to meet deficits by importation. Comparative studies of CVD risk related specifically to milk and dairy products consumption in these regions were not also available at the time of writing this review.

Bovine milk is not a high-fat food and yet due to its considerable SAFA content (72%) [25], its considerable consumption is suspected to lead to higher risk of CVD. The major FAs in bovine milk-fat are palmitic, oleic, stearic, myristic, butyric, lauric, decanoic, and linoleic [25]. Myristic, lauric and palmitic acids are atherogenic and raise the risk of CHD by increasing plasma cholesterol and LDL [26]. However, 18:0 (stearic), 18:1 (oleic) and 18:2 (linoleic acid) reduce the increase [27]. Palmitic acid does not seem to be strongly atherogenic and has not been shown by some workers to raise LDL-C [28], although Grundy [29] showed it potentially raises plasma cholesterol. Although considered high cholesterol foods, milk and dairy products may not be major contributors to dietary cholesterol as whole milk contains 10-15 mg cholesterol/dL [7, 26], unless it is a major item of daily diet as for nomadic pastoralist communities. Moreover, skimming milk to 1% fat reduces cholesterol to <8 mg/dL [7].

The 2000 Kenya annual *per capita* consumption of locally produced beef, goat and lamb, and pork is estimated by this author at 9.4, 1.8 and 0.4 kg, respectively, while that of chicken stood at 1.7 kg. The production and consumption of donkey, camel and game meat in Kenya is undocumented, but potentially represents a small



proportion of the total amount of red meat consumed. The world *per capita* supply of meat was 30 kg as compared to 10 kg for SSA [30].

The per capita fish consumption for the years 2000 and 2001 in Kenya was 7.0 and 5.2 kg, about 19 and 15 g/person/day [30], respectively. Africa's per capita consumption for the year 2005 was 7.0 kg [31]. This is lower than the Japanese per capita consumption of 67 kg [31], which may contribute to the lower prevalence of CVD in Japan [32], although internationalization of Japanese diets may change the scenario in the future. Seafood generally contains 50-80 mg cholesterol/100 g muscle tissue, although shrimp and squid may contain up to 200 mg and >200 mg/100 g muscle tissue, respectively [33]. However, n-3 FAs predominate in the lipids of fish, so that despite the likely considerable cholesterol content, the high degree of unsaturation of fish oils promotes good arterial health. Since most fish oils have >70% PUFA compared to <30% for bovine meat [34], the low saturation minimizes the adverse effects of their considerable cholesterol content. Also, it has recently been postulated that other sterols in fish lipids absorb significant amounts of ingested cholesterol thus lessening its adverse effects. Overall, the long-chain n-3 PUFA, eicosapentaenoic acid (EPA) and docosohexaenoic acid (DHA), commonly found in seafood, are the only FAs that have consistently been shown to significantly reduce the risk of CVD in human kinetic studies [35]. Nevertheless, consumption of excessive amounts of PUFA has been suggested to potentially lead to increased cancer incidence, free radical damage to cells, skin lesions, an increase in serum uric acid, and enhancement of ceroid production.

Animal meats as risk factors for CVD

The general health message to the public about meat consumption is that meat is not good for health because meat is rich in fat and cholesterol, and high intakes are associated with increased blood cholesterol, thereby leading to CHD.

Beef, pork, chicken, and turkey, contain 40-47%, 29-40%, 30-35% and 34-37% SAFA (g/100 g total FAs), respectively [36]. When they contain considerable fat, animal meats can be significant sources of dietary SAFA and cholesterol. Also due to the higher content of cholesterol in organ meats compared to skeletal muscles, it is recommended that their dietary consumption be limited [37]. Untrimmed beef, skinless pork, chicken, and turkey, contain 67-120, 60-67, 78-89 and 78-96 mg cholesterol/100 g edible muscle, respectively.

Game meat tends to have the same amount of cholesterol as beef or pork, but considerably less fat than domestic animal meat [38]. The cholesterol content of both game heart muscle (275 mg/100g tissue) and liver (450 mg/100g tissue) is high [38]. The extent that one should worry about these cholesterol and fat figures depends on how often one eats these organ meats. Some game meat is higher in cholesterol than domestic animal meats, but the combination of more lean body tissue, generally fewer calories, less saturated fat and a significantly higher percentage of cholesterol-reducing PUFA, make game a heart-healthy choice [39]. Game meat also has a significantly higher content of EPA than domestic animal meat. Eicosapentaenoic acid





has been shown to consistently reduce the risk of developing atherosclerosis [35], a major cause of heart attack and stroke. Although the neutral lipids in game meat may be more saturated than those of domesticated animals, phospholipids in game meats such as red deer contained higher PUFA (>55%) [40], than the intramuscular phospholipids in ruminant red meats [41]. Rabbit muscles were also found to have a higher proportion of PUFA (37.4-40.5% of total FAs), with PUFA/SAFA ratios of 0.94-1.02, which were higher than those of pork or chicken [42]. Game meat, therefore, appears healthier than ruminant red meats with respect to CVD risk.

Animal meats are consumed primarily as sources of dietary protein. Raw lean beef, skinless pork, chicken, and turkey, contain approximately 30-35, 20-23, 16-29, and 33-34% protein, respectively. Uncooked lean meat from mule deer, pronghorn antelope, bison, buffalo and the elk generally contain 21-23% protein which compares with the mean protein of 22% in uncooked lean beef [43]. Meat from domestic animals trimmed of visible fat and that from wild life may, therefore, fit into a balanced and low-fat, low-calorie diet that may slow down the development and progression of CVD.

In a recently published review of 54 studies from the literature on red meat consumption as a risk factor for CHD, evidence suggests that lean red meat trimmed of visible fat does not raise total blood cholesterol and LDL-C contrary to popular belief [44]. The studies suggest that lean red meat is low in saturated fat, and if consumed in a diet low in SAFA, it is associated with reductions in LDL-C in both healthy and hypercholesterolemic subjects. Further, lean red meat consumption does not seem to have any effect on *in vivo* and *ex vivo* production of thromboxane and prostacyclins or the activity of haemostatic factors [44]. When the n-3 FA replace primarily the n-6 FA from membrane lipids, the membrane content of arachidonic acid decreases [45]. The result of the decrease in arachidonic acid for eicosanoid formation and competition with the n-3 FAs for the cyclooxygenase and lipooxygenase enzyme sites generally results in decreased production of the 2-series prostanoids, such as thromboxane A_2 (TXA₂) and prostaglandin E_2 . Thromboxane A_2 is a potent stimulus for platelet aggregation and arteriole vasoconstriction [10]. The vascular endothelium, via cyclooxygenase also produces prostacyclin I2 from arachidonic acid, which is a potent antiaggregatory agent and vasodilator of blood vessels [10]. Thus lean red meat, trimmed of visible fat, and consumed in a diet low in saturated fat, does not increase cardiovascular risk factors [44]. However, because animal meats irrespective of their degree of leanness still have considerable amounts of saturated fat and cholesterol, they may still present a risk in regard to CVD morbidity and mortality. Their significance as risk factors for CVD depends on their fat content, the frequency and amount consumed.

Homocysteine (Hcy) is a metabolic intermediate in the biochemical pathway from methionine to cysteine. Despite the seemingly innocuous role, moderately elevated plasma Hcy levels have been shown to be a significant risk factor for CHD [46] and stroke [47], as well as being implicated as a risk factor for Alzheimer's disease [48]. The study by Boushey *et al.* [49] has also provided a strong association between Hcy



ISSN 1684 5374

and CVD. Also, a recent study supports the view indicating that patients with elevated levels of Hcy are roughly 1.7 times more likely to develop coronary artery disease (CAD) and 2.5 times more likely to suffer from a stroke than those with normal levels [50]. However, despite the overwhelming evidence linking Hcy to CVD, questions are also being raised as to whether Hcy is the result of CHD or the cause of it [34]. In a major long-term study spanning 17 years, vegetarians were observed to experience lower death rates from stroke and some cancers than non-vegetarians who regularly consumed meats [51]. The lower death rate in vegetarians from ischaemic stroke may be explained partly by their lower blood pressure and lipid levels, as well as consumption of high-fibre, low-fat plant foods.

Other meat nutrients as risk factors for CVD

Meat is a fair source of the n-3 FAs, a good source of vitamin B₁₂, niacin, zinc and iron. The first three nutrients ameliorate CVD risk, while iron may promote CVD progression. A study of 3,500 individuals established that those consuming <7 mg of dietary zinc/day were more likely to suffer from CAD, diabetes and glucose intolerance [52]. There was a higher incidence of hypertension, high plasma TAG, and lower plasma HDL in the group. Hypertension, hypertriglyceridemia and depressed HDL levels are risk factors for CHD. Also, in the Iowa Women's Study involving 34,492 participants, Lee et al. [53] suggested that a higher intake of haeme iron might be harmful, whereas a higher intake of zinc might be beneficial in relation to CVD mortality in the presence of a trigger that can disturb iron homeostasis, such as alcohol consumption. Meat and liver are the best dietary sources of iron. Liver contains 8.0-25.0 mg iron/100 g of tissue, suggesting caution for lovers of large portions of animal liver, due to its high content of iron, which may promote progression of CVD.

Niacin is commonly used to lower elevated LDL and TAG levels in blood and is more effective in increasing HDL levels than other cholesterol-lowering medications [54]. Studies also suggest that high dose niacin may relieve the symptoms of claudication (difficulty in walking due to cholesteryl deposits in limb joints) [55]. However, clinically beneficial levels of niacin are higher than those in meats, and, are therefore unlikely to be of any significance in CVD amelioration in normal dietary regimes in the absence of supplementation [56]. Linus Pauling Institute [56] suggests the RDA of 16 mg niacin for men and 14 mg for women should be adequate to prevent progression of CVD. Recent studies also show that the combination of niacin and a cholesterol-lowering drug called simvastatin (which belongs to HMG CoA reductase inhibitors or statins), may dramatically slow progression of heart disease, reducing the risk of heart attack, and even death [57].

Fish, a white meat, is an important dietary source of animal protein and the n-3 FAs for fishing communities. Fish oils are rich in the long-chain n-3 FAs, EPA and DHA. These FAs have been found to effectively lower VLDL levels in hyperlipidemic subjects [45]. The average reduction is greater in hypertriglyceridemic subjects than in normolipemic individuals [10]. Apo-protein B-100, an integral part of VLDL synthesis, is also suppressed by the n-3 FAs [10]. Although the overall effects of the





n-3 FAs on serum lipoproteins appear to be beneficial, particularly with respect to VLDL, their susceptibility to oxidation due to their high unsaturation may induce peroxidative damage to LDL, thus increasing the atherogenicity of the lipoprotein fraction [58]. The products of the oxidation of cholesteryl esters, cholestan 3 β , 5 α , 6 β -triol have also been shown to cause endothelial damage both *in vivo* and *in vitro* [59], and may therefore promote atherogenecity. As far as seafood is concerned, most beneficial effects in respect of CVD are likely to be due to the n-3 FAs rather than proteins. The n-3 FAs in fish oils have been shown to reduce arrhythmias, and heart beat rate, and therefore reduce the risk of stroke and sudden death from cardiac arrest [60]. It has also been observed that plasma cholesterol and blood pressure among fisheating populations, are higher than those of vegans, but lower than in consumers of other animal meats. Despite the high cholesterol in seafood, the health benefits of the n-3 FAs on the cardiac system seem to outweigh risks resulting from the considerable cholesterol content.

Dietary protein is generally considered to be of minor importance in the aetiology of CVD. However, epidemiological studies have shown the consumption of animal protein is positively correlated with CVD incidence, while vegetable protein is negatively correlated with CVD mortality rates. Although animal studies have shown animal protein as being more atherogenic than vegetable protein, it is more difficult to factor out these effects in human studies [11].

CONCLUSIONS

Although dietary protein is of minor importance in the aetiology of CVD, epidemiological studies have shown consumption of animal protein is positively correlated with CVD incidence, while that of vegetable protein is negatively correlated with CVD mortality rates. When they contain considerable fat, animal meats can be significant sources of dietary saturated fat and cholesterol. Due to the higher cholesterol content of organ meats compared to skeletal muscles, it is recommended that their dietary consumption be limited. Because animal studies have generally shown animal protein as being more atherogenic than vegetable protein, it seems beneficial to reduce the dietary intake of SAFA and cholesterol from meats and other high-fat, high-cholesterol animal foods, since reduction in serum total cholesterol results in reduction of coronary morbidity and mortality. This review, therefore, recommends reduction in the dietary intake of fatty red meat, while promoting consumption of lean white meats.

REFERENCES

- 1. **American Heart Association** Heart attack and signs of stroke. American Heart Association, Washington, D.C. 2008. Found at: <u>http://www.americanheart.org/</u>. Accessed on 15th April 2008.
- 2. Vasan RS, Larson MG, Leip EP, Evans SC, O'Donnell CJ, Kannel WP and D Levy Impact of high blood pressure on the rise of CVD. *New Eng. J. Med.* 2001; **45**: 1291-1297.
- 3. **WHO** (World Health Organization) WHO cardiovascular diseases Mortality estimates. World Health Organization, Geneva, 2005. Found at: http://www.who.int/. Accessed on 20th August 2007.
- 4. Matthews KA, Meilahn E, Kuller LH, Kelsey SF, Caggiula AW and RR Wing Menopause and risk factors for coronary heart disease. *New Eng. J. Med.* 1989; **321(10)**: 641-646.
- 5. **Murray CJL** and **AD Lopez** Mortality by cause for 8 regions of the World. Global burden of disease study. *Lancet* 1997; **349**: 1269-1276.
- 6. **American Heart Association** Heart attack and related diseases. American Heart Association, Washington, D.C. 2004. Found at: <u>http://www.americanheart.org/</u>. Accessed on 15th August 2007.
- 7. **Myant NB** The Biology of Cholesterol and Related Steroids. William Heinemann Medical Books Ltd., London, 1981: 126, 133.
- 8. American Heart Association Nutrition Committee AHA Dietary Guidelines. Revision 2000: A Statement for Healthcare Professionals from the Nutrition Committee of the American Heart Association. *Circulation* 2000; **102**: 2284–2299.
- 9. Emholm C, Huttunen JK and P Pietinen Effect of diets on serum lipoproteins in a population with a high risk of coronary heart disease. *New Eng. J. Med.* 1982; **307**: 850-855.
- 10. **Bruckner G** Fatty acids and cardiovascular diseases. In: Chow CK(Ed.). Fatty Acids in Foods and their Health Implications, 2nd edn. Marcel Dekker, New York and Basel, 2000: 843-863.
- 11. **Kris-Etherton PM** and **S Yu** Individual fatty acids effect on plasma lipids and lipoproteins: Human studies. *Am. J. Clin. Nutr.* 1997; **65**(Suppl): 1628S-1644S.
- 12. Bonow RO, Smaha LA, Smith SC Jr., Mensah G and C Leufant The international burden of cardiovascular disease. Responding to the emerging global epidemic. *Circulation* 2002; **106**: 102.



- 13. Swai AB, Mclarty DG, Kitange HM, Kilima PM, Tatalla S, Keen N, Chuwa LM and KGMM Alberti Low prevalence of risk factors for coronary heart disease in rural Tanzania. *Int. J. Epidemiol.* 1993; **22**(4): 651-659.
- 14. Edwards R, Unwin N, Mugwusi F, Whiting D, Rashid S, Kissima J, Aspray T, and KGMM Alberti Hypertension prevalence and care in an urban and rural area of Tanzania. *J. Hypertension* 2000; **18**(2): 145-152.
- 15. **Kurjiwala RL** Some reflections on milk supply and consumption statistics in Tanzania with particular reference to the role of the traditional cattle herds. In: the livestock sector and human nutrition demand in Tanzania. Proc. 15th Sci. Conf. Tanzania Soc. Anim. Prod. 1988; **15**: 216-242.
- 16. **Massae EE** Experience with dairy development in Tanzania. In: Seminar Proc. Dairy Dev. Policy and Implementation. Sharing experiences between Africa and Asia. Harare, Zimbabwe, 12-16th July 1993.
- 17. Van der Sande MAB, Robin B, Faal H, Banya WAS, Dolin P, Nyan OA, Ceesay SM, Walraven GEL, Johnson GJ and KPWJ McAdam Nationwide prevalence study of hypertension and related non-communicable diseases in the Gambia. *Trop. Med. Int. Health* 1997; **2**(11): 1039-1048.
- 18. **FAO.** Fishery statistics. FAO, Rome 1995. Found at <u>http://www.fao.org</u>. Accessed 14th September, 2009.
- 19. **Gambia Agricultural Service** Food Security in the Gambia. Government of Gambia, Libreville, 2007. Found at: <u>http://www.statehouse.gm/agriculture/achievements, 94-to_date.htm</u>. Accessed on 14th September. 2009.
- 20. Anabwani GM and P Bonhoeffer Prevalence of heart disease in school children in rural Kenya using colour-flow echocardiography. *East Afr. Med. J.* 1996; **73(4)**: 215-217.
- 21. Kengne AP, Amoah AGB and JC Mbanya Cardiovascular complications of diabetes mellitus in sub-Saharan Africa. *Circulation* 2005; **112**: 3592-3601.
- 22. Steyn K, Sliwa K, Hawken S, Commerford P, Onen P, Damasceno A, Ounpuu S and S Yusuf Risk factors associated with myocardial infarction in Africa. The Interheart Africa study. *Circulation* 2005; **112**: 3554-3561.
- 23. Lokuruka MNI Role of fatty acids of milk and dairy products in cardiovascular diseases: A Review. *AJFAND* 2007; 7(1): 45-59.
- 24. FAO. State of Fisheries and Aquaculture (SOFIA). FAO, Rome, 2008.

Pablished by African Scholarfy Science ConvenzesionHone Trant Josem Trant Plane, Buryala Road, Upper Hill, Nairobi P.O. Box 29086-60025 Tel +254-20-2351785 Fax: +254-20-4444030, Nairobi, KENYA Enail: enaigeo()iscenses: co.lor. OR: info)(agfined ant www.ajfand net

- 25. **Posati JP**, **Kinsella JE** and **BK Watt** Comprehensive evaluation of fatty acids in foods. 1. Dairy products. *J. Am. Diet. Assoc.* 1975; **66**: 482-488.
- 26. **Jensen RG** Fatty acids in milk and dairy products. In: Chow CK (Ed.). Fatty Acids in Foods and their Health Implications, 2nd edn. Marcel Dekker, New York and Basel, 2000: 109-124.
- 27. **Miller GD**, **Jarvis JN** and **LD McBean** Dairy Foods and Cardiovascular Health. In: Miller GD, Jarvis JN and LD McBean (Eds.). Handbook of Dairy Foods and Nutrition. CRC Press, Boca Raton, Florida, 1995: 1-37.
- 28. McNamara DJ Effect of Fat-modified Diets on Cholesterol and Lipoprotein Metabolism. *Ann. Rev. Nutr.* 1987; **7**: 273-290.
- 29. Grundy SM Comparison of monounsaturated fatty acids and carbohydrates for lowering plasma cholesterol. *New Eng. J. Med.* 1986; **314**: 745-748.
- 30. **FAO**. 2004 Fisheries data. FAO Rome, 2004 February. Found at: <u>http://faostat.fao.org/faostat/collections?version=ext&hasbulk=0&subset=fisheri</u> <u>es</u>. Accessed 11th Jan 2008.
- 31. Food and Agriculture Organization FAO Yearbook. Fisheries Statistics and Commodities. FAO, Rome, 2005; **81**: 11, 13, 46, 71, 74, 169-172.
- 32. Ascherio A, Rimm, EB, Stampfer EJ, Giovannucci EL and WC Willett Dietary intake of marine n-3 fatty acids, fish intake and the risk of coronary disease among men. *New Eng. J. Med.* 1995; **332**: 977-982.
- Holland B, Brown J and DH Buss Fish and Fish Products; the Third Supplement to McCance & Widdowson's The Composition of Foods (5th edn.), HMSO, London. 1993.
- 34. **Pryer J** and **P** Shetty Adult nutrition. In: Shetty P (Ed.). Nutrition through the Life Cycle. Leatherhead Publishing, Surrey, and Royal Society of Chemistry, Cambridge, UK, 2002: 91-117.
- 35. **ISSFAL** (International Society for the Study of Fatty Acids and Lipids) Recommendations for Intake of Polyunsaturated Fatty Acids in Healthy Adults. International Society for the Study of Fatty Acids and Lipids Board, 2004.Found at <u>www.issfal.org/</u>. Accessed on April 16, 2008.
- 36. **USDA.** Composition of foods: fats and oils-raw, processed, prepared. Agriculture Handbook No. 8-4, USDA Consumer and Food Economics Institute, Washington, D.C., 1979a.
- 37. USDA-USDHSS. Nutrition and your health: dietary guidelines for Americans. USDA Home and Garden Bull. No. 232, 1985.

Published by African Scholarly Science Communications Trust Josem Trust Place, Buzyska Road, Upper Hill, Narobi P.O. Box 29086-00625 Tel: +254-20-2351785 Fax: +254-20-4444030, Narobi, KENYA Email: enlange@sceneet.co.ke_OR_info@agfand.net_____www.ajfand.net______

- 38. **Brown L** (Ed.) Fat and cholesterol content of wild game. New release archive, Penn State's College of Agricultural Sciences, 2005.
- 39. North Dakota University and USDA Comparative Nutritional Table: Calories, cholesterol, fat, and protein content of various types of meat (3-ounce cooked portions). North Dakota University Extension Services, Grand Forks, North Dakota, 2005.
- 40. **Manley TR** and **DA Forss** Fatty acids of lipids from young deer. J. Sci. Food Agric. 1979; **30**: 927.
- 41. Allen CE and EA Foegeding Some lipid characteristics and interactions in muscle foods-a review. *Food Technol*. 1981; **35**(5): 253.
- 42. Griffiths TW, Gandemer G, Viau M and P Vedrenne Polyunsaturated fatty acid (PUFA) content of rabbit meat: a potential source of PUFA for human nutrition. *Proc. Nutr. Soc.* 1989; **48**: 5A.
- 43. Medeiros LC, Busboon JR, Ray A, Field J, Williams C, Miller GJ and B Holmes B-920R-Nutritional Content of Game Meat. Departments of Family and Consumer Sciences and Animal Sciences Cooperative Extension Service, College of Agriculture, University of Wyoming. 2005.
- 44. Li D, Wahlqvist ML, Mann NJ and AJ Sinclair Lean meat and heart health. *Asia Pac J. Clin. Nutr.* 2005; **14**(2):113-9.
- 45. **Harris WS** n-3 fatty acids and serum lipoproteins: human studies. *Am. J. Clin. Nutr.* 1997a; **65**(Suppl): 1611S-1616S.
- 46. Graham I Homocysteine in health and disease. Ann. Intern. Med. 1999; 131: 387-8.
- 47. Bazzano LA, Jiang He, Ogden LG, Loria C, Vupputuri S, Myers L, Whelton PK and SE Kasner Dietary intake of folate and risk of stroke in US men and women: NHANES I epidemiologic follow-up study. *Stroke* 2002; 33: 1183-89.
- 48. Loscalzo J Homocysteine and dementias. New Eng. J. Med. 2002; 346: 466-8.
- 49. **Boushey CJ, Beresford SA, Omenn GS** and **AG Motulsky** A quantitative assessment of plasma homocysteine as a risk factor for vascular disease: probable benefits of increasing folic acid intakes. *JAMA* 1995; **274**:1049-57.
- 50. Cleophas TJ, Hornstra N, van Hoogstraten B and J van der Meulen Homocysteine. A risk factor for coronary artery disease or not? A metaanalysis. *Am. J. Cardiol.* 2000; **86**:1005-9.

Published by African Scholarly Science Communications Trust Joern Trust Planc, Burysia Road, Upper Hill, Nairobi P.O. Box 29086-00025 Ye1 +254-20-2351718 Fan: +254-20-4444030, Nairobi, KENYA Brasil: maiage/disconsect.co.lo: OR: http://githud.act www.ajfand.act



- 51. Key TJA, Thorogood M, Appleby PN and ML Burr Dietary habits and mortality in 11,000 vegetarians and health conscious people. Results of a 17-year follow-up. *BMJ* (British Medical Journal) 1996; **313**: 775-779.
- 52. Singh RB, Niaz MA, Rastogi SS, Bajaj S, Gaoli Z and Z Shoumin Current zinc intake and risk of diabetes and coronary artery disease and factors associated with insulin resistance in rural and urban populations of North India. *J. Am. Coll. Nutr.* 1998; **17**(6): 564-570.
- 53. Lee DH, Folsom AR and DR Jacobs, Jr Iron, zinc, and alcohol consumption and mortality from cardiovascular diseases: the Iowa Women's Health Study. *Am. J. Clin. Nutr.* 2005; **81(4)**: 787-791.
- 54. **Guyton JR** Effect of niacin on atherosclerotic cardiovascular disease. *Am. J. Cardiol.* 1998; **82**: 18U–23U.
- 55. **O'Hara J** and **CG Nicol** The therapeutic efficacy of inositol nicotinate (Hexopal) in intermittent claudication: a controlled trial. *Br. J. Clin. Prac.* 1988; **42(9)**: 377-381.
- 56. **Linus Pauling Institute** Niacin. The Linus Pauling Institute, Oregon University, Corvallis, USA, 2002.
- 57. Brown BG, Zhao XQ, Chait A, Fisher LD, Cheung MC, Morse JS, Dowdy AA, Marino EK, Bolson EL, Alaupovic PJF, Serafini L, Ellen HF, Wang S, DeAngelis D, Dodek A and JJ Albers Simvastatin and niacin, antioxidant vitamins, or the combination for the prevention of coronary disease. *New Eng. J. Med.* 2001; 345(22): 1583-1592.
- 58. **Tsai PJ** and **SC Lu** Fish oil lowers plasma lipid concentrations and increase the susceptibility of low density lipoprotein to oxidative modifications in healthy men. *J. Formos. Med. Assoc.* 1997; **96(9)**: 718-726.
- Hennig B and GA Boissonneault Cholestan 3β, 5α, 6β-triol decreases barrier function of cultured endothelial cell monolayers. *Atherosclerosis* 1987; 68: 225-261.
- 60. Geelen A, Brouwer IA, Schouten EG, Maan AC, Katan MB and PL Zock Effects of n–3 fatty acids from fish on premature ventricular complexes and heart rate in humans. *Am. J. Clin. Nutr.* 2005; **81(2)**: 416-420.